Heart Failure

Heart failure: progressive disorder in which the heart is unable to pump sufficient blood to meet the needs of the body.

Clinical presentation:
A-dyspnea (pulmonary oedema)

B-lower limb oedema and liver enlargment(congestion)

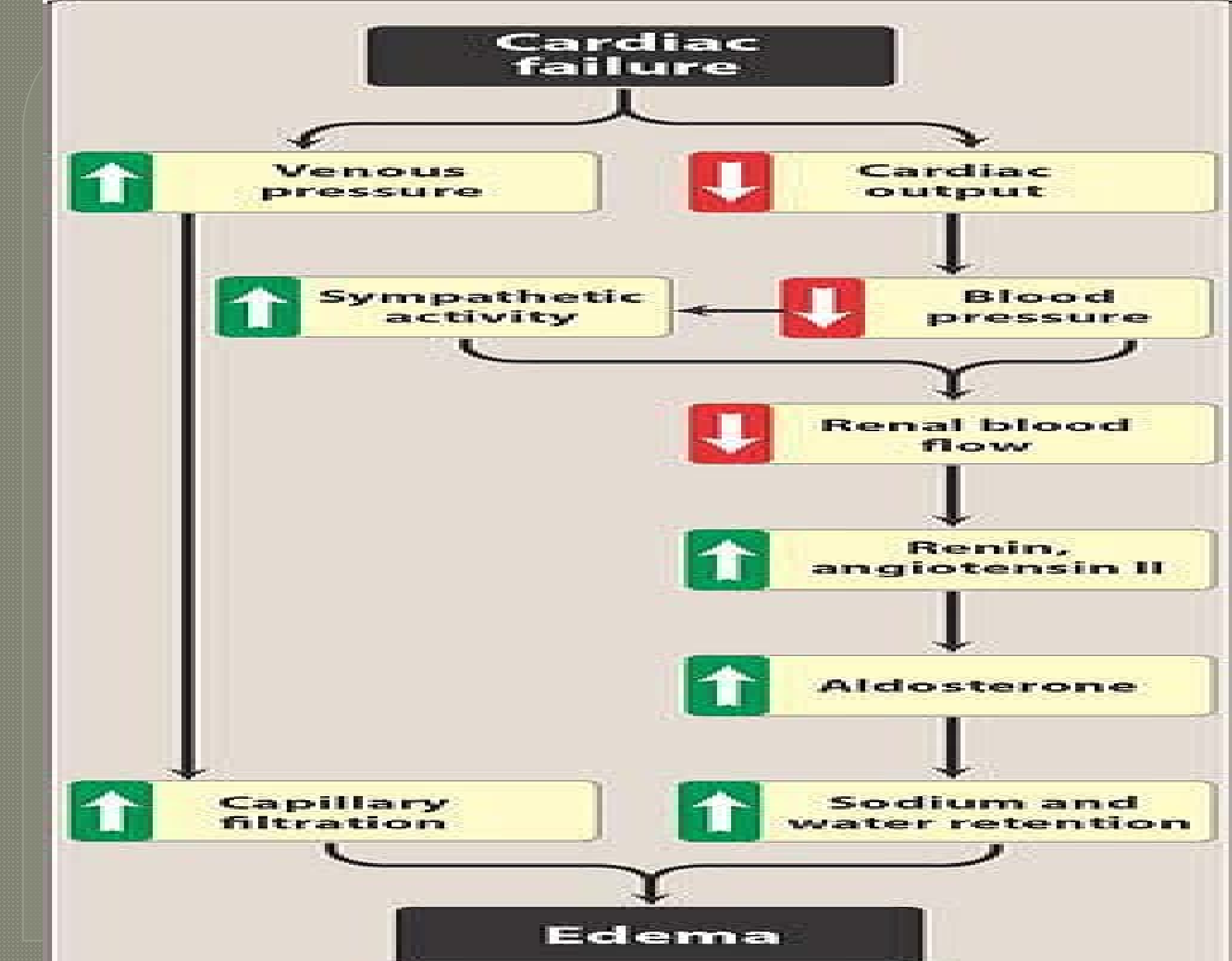
C-Fatigability (hypoxia)

Causes:
A- ischemic heart disease

B-hypertension

C-valvular lesions

D-cardiomyopathy.

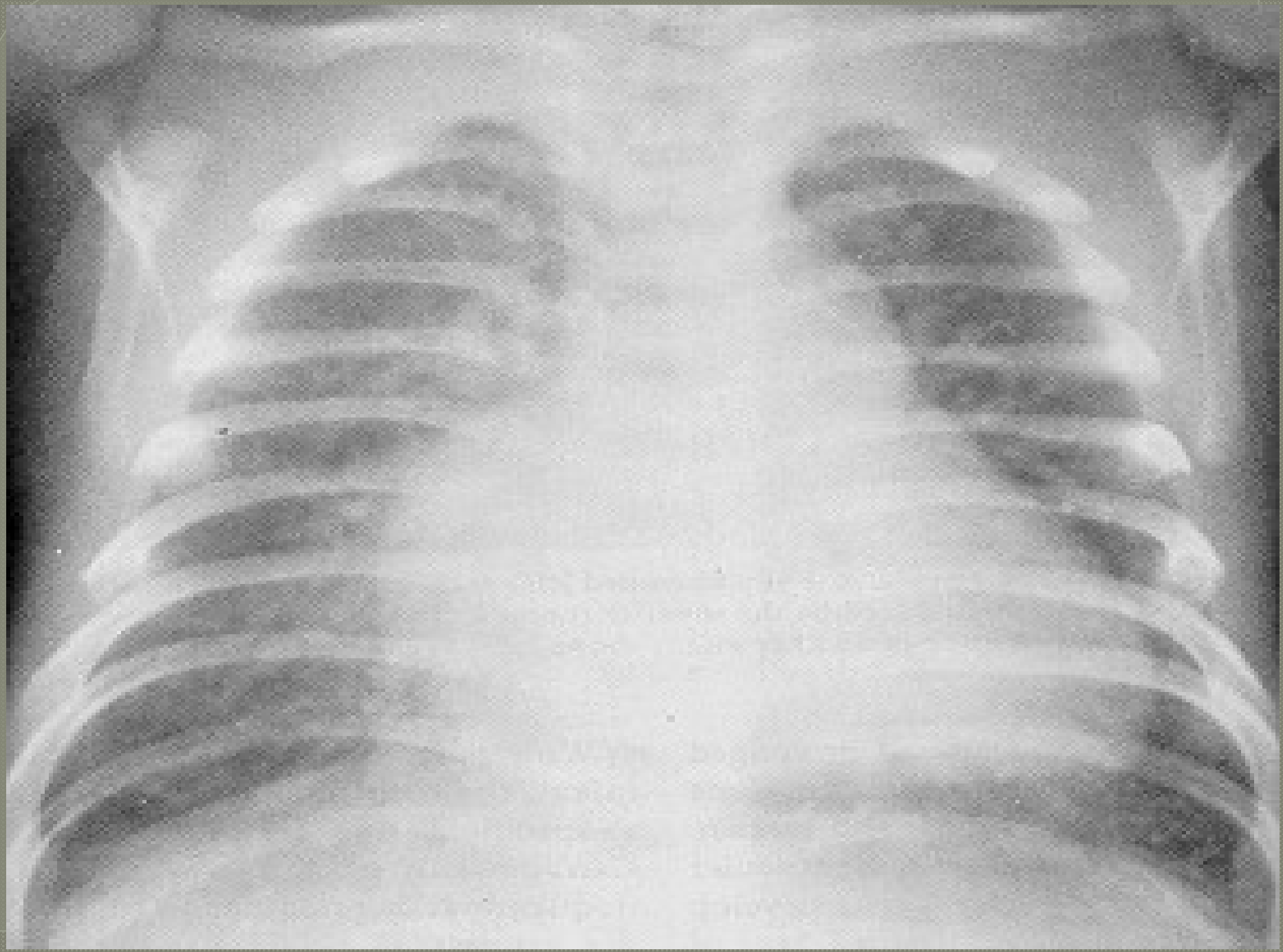

Cardiac failure
Venous pressure
Cardiac output
Sympathetic activity
Blood pressure
Renal blood flow
Renin, angiotensin II
Aldosterone
Capillary filtration
Sodium and water retention
Edema

Compensation of heart failure:

A- sympathetic overactivity due to low blood pressure

B-renin angiotensin system stimulation due to renal low perfusion

C- hypertophy and diltation of cardic muscles



Mangment of heart failure:

A- bed rest.

B- low salt intake.

DRUGS USED in mangment of heart failure:

A-**Diuretics**

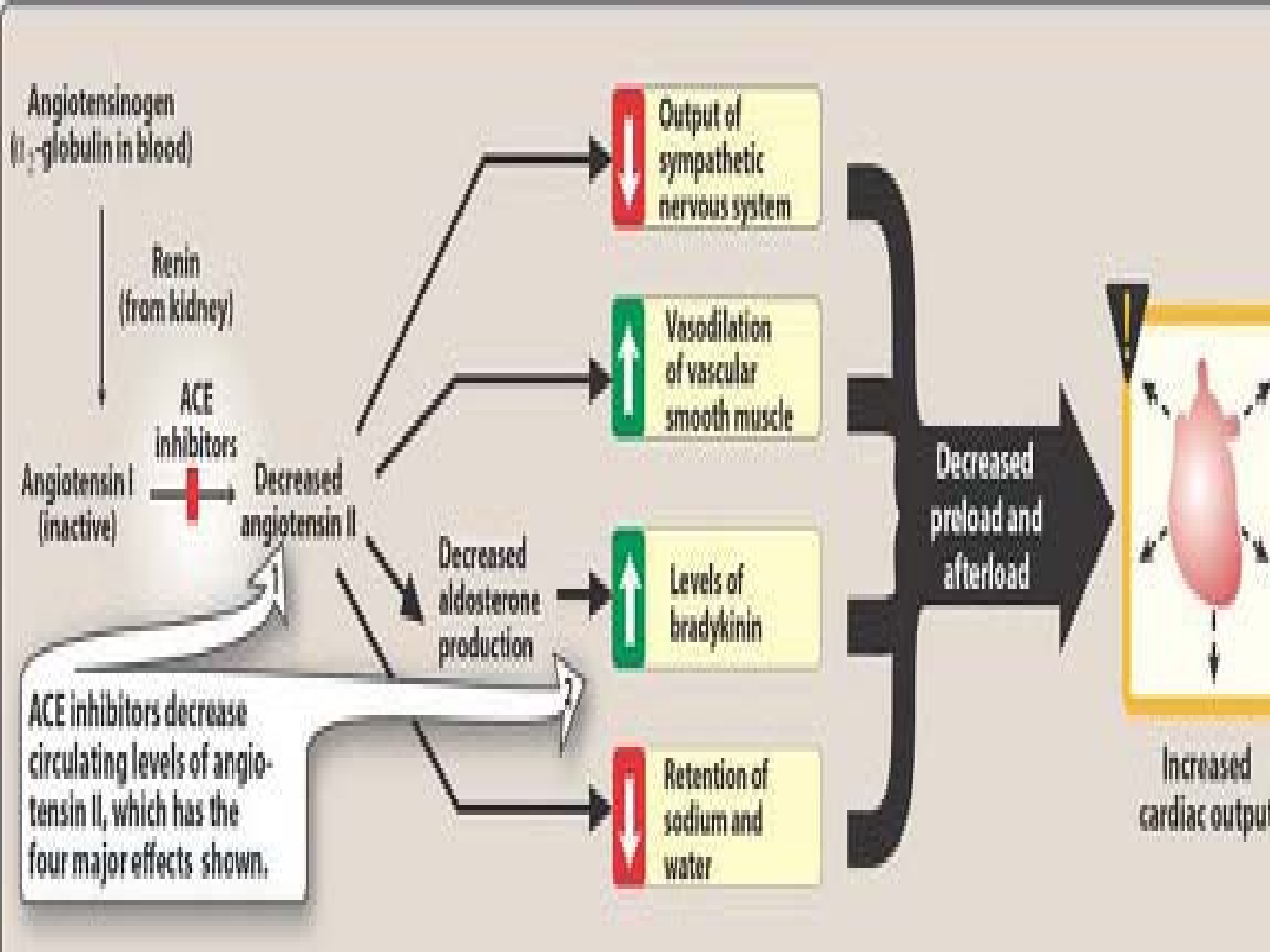
Diuretics relieve pulmonary congestion and peripheral edema. These agents are also useful in reducing the symptoms of volume overload, including orthopnea and paroxysmal nocturnal dyspnea. Diuretics decrease plasma volume and, subsequently, decrease venous return to the heart (preload

Example ; loop diuretic (fursamide)

Common side effect: hypovolemia and hypokalemia.

B- vasodilator agent: ACEI OR ARBS.

ACEI used in HF started with low dose , to prevent hypotension .


Angiotensinogen
(α_2-globulin in blood)
Renin
(from kidney)
ACE
inhibitors
Angiotensin I
(inactive)
Decreased
angiotensin II
Decreased
aldosterone
production
Output of
sympathetic
nervous system
Vasodilation
of vascular
smooth muscle
Levels of
bradykinin
Retention of
sodium and
water
Decreased
preload and
afterload
Increased
cardiac output
ACE inhibitors decrease circulating levels of angiotensin II, which has the four major effects shown.

C- beta blckers: reduce mortality in patients with HF but contraindicated in acute severe HF.

Start beta blocker in stable heart failure.

Beta blockers used in heart failure:

1-carvidolol (non selective alpha and beta)

B- metoprolol (selective beta 1 blockers)

C-bisoprolol (selective beta 1 blockers)

Several studies demonstrate beta blockers .improve systolic function and reversed remodelling result in reduce mortality.

IN Pt not respnse to loop duiretic , ACEI and beta blocker can used spironlactone or digoxin.

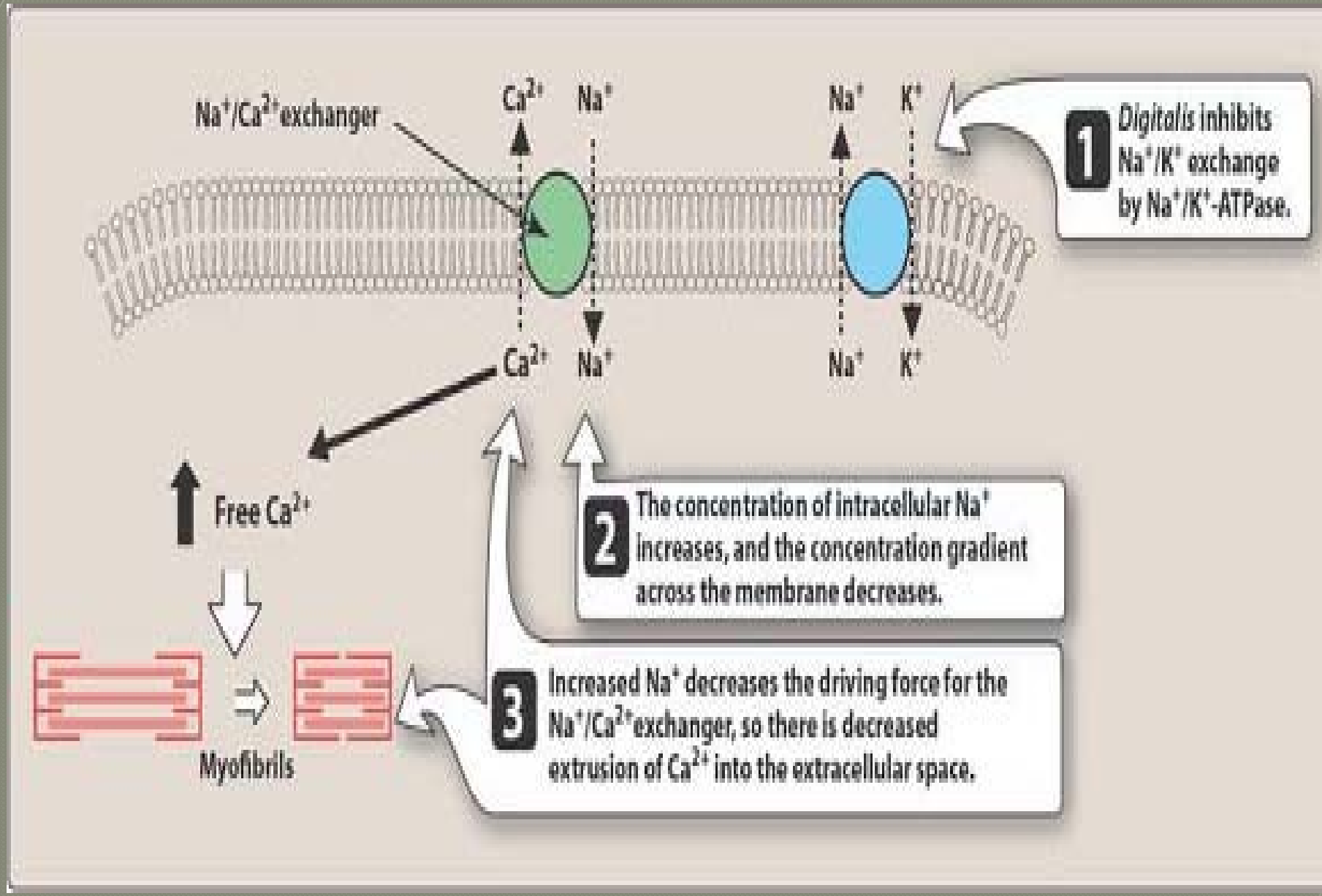
Pt with contraindiction of ACEI can used combination nitrate + hydralzine.

D. Inotropic Drugs:

Digitalis(digoxin).

Mechanism of action

Block Na+ ,k+ pump.lead to increase Ca+ inside the cardic muscle result in increase contractility


Na+/Ca2+exchanger
Ca2+
Na+
Na+
K+
1 Digitalis inhibits Na+/K+ exchange by Na+/K+-ATPase.
Ca2+
Na+
Na+
K+
Free Ca2+
2 The concentration of intracellular Na+ increases, and the concentration gradient across the membrane decreases.
3 Increased Na+ decreases the driving force for the Na+/Ca2+exchanger, so there is decreased extrusion of Ca2+ into the extracellular space.
Myofibrils

Also increase vagus tone result in decrease heart rate.

CLINCAL use:

A- HF with atrial fibrilation

B- HF not response to conventional treatment.

Pharmacokintic:
oral or IV.
Half life: 36 hours.

Narrow margin of safety.

Larg volume of ditrubution.
elimination by renal

Side effect:

- **cardiac effects: The common cardiac side effect is arrhythmia**

- **Gastrointestinal effects: Anorexia, nausea, and vomiting are early sign of toxicity.**
- **Central nervous system effects: These include headache, fatigue, confusion, blurred vision, alteration of vision(yellow vision)**

- Severe toxicity
- resulting in ventricular tachycardia may reqiure use of antibodies to *digoxin (digoxin immune Fab), which bind and inactivate the drug.*

Factor increase toxicity is low K+ and verapamil.

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